British Journal of Anæsthesia

Vol. XXIV, No. 1 January 1952

EDITORIAL

In a previous Editorial published in this Journal (1951, Vol. 23, No. 2) we expressed concern at the perpetuation of a second grade of anæsthetic specialists. It must be the unanimous opinion of those involved in this specialty that anæsthesia demands practitioners with the complete background and training necessary for Consultant status. This is implied in the very nature of the work and in the degree of clinical responsibility undertaken by anæsthetists. Nevertheless, the Ministry of Health, in its circular RHB(50)96, expressed the view that "there will often be a need in some hospitals for Assistant Anæsthetists remunerated on the senior hospital medical officers scale". Just how often and under what circumstances has never been defined. The result is that the interpretation of this regulation and consequently the status and terms of service of anæsthetists vary greatly from region to region. It would appear that some regions consider that the less important hospitals on the periphery should be staffed with Assistant Anæsthetists, and such appointments are being made with a frequency which, although it may be financially expedient, would indicate very little consideration of the principles or issues involved.

The representative bodies of anæsthetists in this country have, so far, served the interests of the specialty well by

ensuring more or less equality of status with the other specialties. A little thought, however, will reveal that in this tendency to the haphazard and ill-considered establishment of Assistant Anæsthetist posts, much of their good work may be undone. It is of grave concern to all with the interests of anæsthesia at heart that these bodies should observe this evolution carefully and see that the prestige and greatness of British anæsthesia are not undermined.

It is our boast that in this speciality we and the United States lead the world and, when the state of affairs in other countries is viewed, one realizes how much they depend on us for assistance and support in the development along the right lines of their anæsthetic services. We have a duty not only to ourselves, but also to them, and one has confidence that those in this country who have the interests of the specialty at heart, and who have the power to influence events, will continue in their efforts to ensure that these high standards in work and personnel are maintained.

Brit. J. Anæsth. (1952), **24**, 3.

CHEST MOVEMENTS AND THE INTERCOSTAL MUSCLES*

By W. B. PRIMROSE

To the anæsthetist, no bodily movement is so important as that exhibited by the breathing, as it provides information concerning the condition in which the unconscious patient may be at any particular time and also signs by which the administration of the anæsthetic has to be regulated. This being so, it is strange that of the many efforts to analyse chest movement, none seems to have given complete satisfaction, and the matter is to-day still in the position described by McKendrick in 1888 as " very confused ". Robert Gesell (1936), in a more recent work on the respiratory muscles, indicates this confusion with reference to the intercostal muscles alone, and the subjoined list given by him after Luciani (1911) is worthy of note.

(*a*) Both external and internal intercostal muscles are inspiratory. (Borelli, Senac, Boerhaave, Winslow, Haller, Cuvier, Duchenne.)

(*b*) Both kinds of muscle are expiratory. (Vesalius, Diemerbrock, Sabatier, Beau and Maissac, Longet.)

(*c*) The external intercostals are inspiratory, the internal are expiratory with the exception of the intercartilaginous portions. (Spigel, Vesling, Bayle, Hamburger, Hutchison, A. Fick, Martin and Hartwell.)

* Condensed from a Presidential Address given to the Glasgow and West of Scotland Association of Anæsthetists, March 22nd, 1951 ; also from a paper read to the Anæsthetic Section of the Royal Society of Medicine on January 4th, 1952.

The writer wishes to acknowledge with gratitude the facilities provided by Professor Garry of the Institute of Physiology in the University of Glasgow for the experiments described ; also the assistance given by members of his staff.

(*d*) The intercostals are of no great importance in regard to the movements of the ribs: they serve rather to regulate the tension in the intercostal spaces, and to reinforce them during inspiration, impeding their retraction by the increased negative intra-thoracic pressure. (Henle, Meissner, Brucke, von Ebner, Landois.)

To account for such diversity of view, it is necessary to examine the interpretation of chest movement as given by the anatomist, since this has always been accepted as sound, certainly as reasonable, and has always been taken as the starting point of the many physiological researches into the problem of chest movements.

From comparative studies of body-wall structure, the anatomist has been led to believe that the bilaminar intercostal muscles, being so similar in appearance and relationship to the bilaminar series of such simple forms as the earthworm, operate in a similar manner, that is, that the two muscular layers alternate and oppose each other in action and so account for the two phases of breathing. The fact was never noted, however, that while the physical conditions presented by the worm demanded the opposing actions of a circular and a longitudinal body-wall musculature, those same conditions were not present in higher cœlomate forms in which a different body-wall physiology obtained. In these, although a laminated system of muscles was also required, it had to carry out quite different functions. The failure to note this difference of function arose from the devotion of morphologists to the idea of metameric segmentation of the body. This is a scheme or plan based entirely upon appearances presented by serially repeated parts in the so-called segmented cœlomate animals, and from want of demonstration of the actual existence of the condition, the idea has now fallen considerably into disfavour, especially as its use in express-

ing fundamental body structure has led to confusion in this problem of chest movements and others also. With this in view, the approach to the understanding of chest movements must be made from direct observation uninfluenced by any artificial preconceptions which have never been able to demonstrate actual function.

When radical mastectomy was commonly performed for cancer of the breast, the exposed chest provided excellent opportunities for close observation of the external intercostal muscles as these were always undisturbed. Two movements were apparent, namely, the to-and-fro movement of the chest as a whole, and frequently, the insucking of the intercostal muscles with each inspiration. These intercostal muscles appeared to offer little resistance to the negative intrapleural pressure set up by the descending diaphragm, and they certainly did not appear to contract rhythmically as was shown if some of the fibres were accidentally cut: the incision so made merely gaped without rhythmical alteration, indicating a state of tonic contraction of no great power or apparent purpose.

A further observation made in the course of some cerebellar operations was the contraction of the cut ends of the reflected occipital muscles during inspiration. In this operation the patient was lying prone and unconscious. The muscles, epimeric according to anatomical grouping, seemed to be strange associates in the respiratory act which is carried out by hypomeric muscles normally. This served to show that breathing, rendered difficult by the prone position, could call into play any muscles that could exert any influence upon the chest, however indirectly, and quite independently of any morphological arrangement. It also presented clear *visible* evidence of muscular contraction about which there could be no doubt. When, from

evidence of fatigue in these long operations, the patient was suitably supported, giving the diaphragm unrestricted freedom in its descent, the occipital muscles no longer acted in this way.

It has also often been noticed that when artificial respiration has been called for in the course of anæsthetic practice, that the physical effort required in this procedure was out of all proportion to the effects produced, particularly in elderly subjects. This seemed to show that the chest tended to fixation and did not contribute greatly to breathing, and this in spite of a freely movable system of diarthrodial joints between it and the vertebral column.

These observations stimulated interest in the motive power exercised upon the chest, especially as the intercostals did not appear to provide any; and, as other experimenters have found, the problem is centred upon these muscles which all have desired to see actually working.

With this end in view, a beginning was made by the direct observation of the breathing muscles of a dog—greyhound—and of those other body-wall muscles considered to be related to breathing. Recalling the visible contraction of the cut ends of the occipital muscles in the above-mentioned cerebellar cases, similar contraction was looked for by cutting the fibres of the various muscles. No action of any kind could be found, even when the fibres were examined under some magnification (20 diams.). The intercostals, which in the greyhound are well developed, gave no indication of any contractile effort although the chest exhibited its normal to-and-fro movements. It was also not possible to discern any movement of the ribs, each to each, for in spite of skeletal limitations they are permitted a small degree of independent movement which their intrinsic muscles would show if they acted with any

vigour. With the exception of the diaphragm, which was obviously in action, the same was observed with regard to all the other muscles examined in this way. Even the sterno-mastoids, which could be the most powerful elevators of the chest, were quite inactive, and the abdominal muscles did nothing to retract the chest. The negative findings of this simple examination seemed to show that none of the body-wall muscles credited with respiratory function from their anatomical associations with the chest appeared to act with any physiological purpose.

An investigation with a similar purpose was carried out by Sharpey Schafer and MacDonald in 1925, but it was combined with the use of the string galvanometer to detect muscle potentials. While the latter part of the experiment was not successful, the observations given in the account are of interest.

These workers isolated a group of three ribs with their intercostal muscles from the sternum and from the adjacent ribs by section of the intercostal muscles before and behind and from sternum to vertebral column. They recorded that the isolated group kept pace with the rest of the chest and they were at a loss to account for this, since they expected that the external intercostals would show evidence of contraction in advancing the ribs with inspiration and that the internal muscles would show the reverse movement. Nothing like this seems to have happened, for the paper ends with these words: "We had supposed it would be easily possible to resolve this question of the active participation of the internal intercostals in the expiratory movement of the ribs by the employment of the string galvanometer, but have met with unexpected difficulties in applying that method, and have not yet succeeded in

entirely overcoming these difficulties." To these experimenters, the difficulties of assessing costal movement have arisen from two sources. In the first instance, they did not succeed in obtaining visible contraction of the muscles; and secondly, they were dominated by the anatomical convention which required that these muscles oppose each other in action to account for inspiration and expiration as separate muscular acts.

This experiment, in spite of its unsuccessful issue, appeared to be worthy of repetition, but with some modification. It seemed that if the ribs exhibited gross movement as a chest, then that movement could be stopped by section of muscles other than the intercostals, since these had obviously failed to influence the movement of the ribs in the above experiment. It was decided to follow this line of investigation.

For this experiment rabbits were used: they were intubated through tracheotomies for control of respiration and also anæsthesia. The ribs of one side, the left, were exposed with their intercostals and detached from the sternum, with the exception of the first, in order to avoid damage to the subclavian vessels. This was done by cutting through the costal cartilages close to the side of the sternum so that these ribs would have no contact with that bone. Isolation of the group of ribs was completed by section of the first intercostal muscles from front to back.

Arranged in this way, the experimental ribs with their intercostal muscles moved to-and-fro, keeping pace with the sound side, although having no contact with it. The pectoral and the serratus muscles were reflected and the abdominal muscles were detached from the lower costal margin, as was also the diaphragm. These sections, however, made no difference, the ribs continuing to move

in perfect order as a chest. Finally, the intercostals were cut from front to back as far as the *angles* of the ribs. The normal uniform movement of the ribs continued, and their *forward* thrust could be distinctly felt as before. This seemed to point clearly to the fact that the intercostals had little to do with the visible movements of normal breathing. Some other muscular agency was providing the power.

Another rabbit was prepared in the same way so as to display the intercostal muscles; but this time the dorsal spinal muscles having attachments to the ribs were removed. It was desired to expose the *levatores costarum* as being the muscles most closely identified with the ribs and the vertebral column. On finding and dividing these, the ribs and their intact intercostal muscles at once came to rest. This seemed rather surprising, knowing that our own levatores costarum are small and feeble muscles quite incapable of producing this degree of costal movement. In fact, Keith (1903) regards them as acting upon the vertebral column. Enquiry at the Department of Anatomy, Glasgow Veterinary College, revealed the fact that in all the veterinary mammals the levatores costarum are relatively large and fleshy in the adult.

> This statement is in keeping with that of Markwald (1888), who, writing on movements of respiration in the rabbit, stated that the diaphragm is not essential to life in adult rabbits, as these show a considerable degree of thoracic breathing. Rabbits under four to six months of age, however, die as they have not gained the full use of costo-vertebral muscles.

A peculiar and most fortunate accident occurred during this experiment. The tracheal cannula proved to be a trifle too large and the trachea split slightly. This necessitated the use of a smaller cannula, which was inserted, but it proved to be inadequate for normal breathing and so set

up dyspnœa. The effect of this upon the muscles that were being examined was remarkable. The intercostals, which had always been at rest, now contracted with great vigour, pulling the ribs strongly together with each inspiration and allowing them to relax with each expiration. Along with this activity, the infra-hyoids and pectoralis minor muscles were seen to be in active and simultaneous contraction. This may also have included the scalene, which is single in the rabbit, but this was not actually observed. MacKenzie (1919) believes that this muscle acts upon the neck. Of the other muscles, sterno-mastoid, pectoralis major, serratus anterior and the abdominal group, no sign of activity presented itself even under these conditions of urgency.

Reviewing these experiments briefly, the first rabbit showed clearly that the ribs, free to move, were not being acted upon by their intercostal muscles, otherwise they would have been crowded together by the external intercostals on inspiration. There was also no tendency for them to be actively separated by any muscles, least of all by the internal intercostals, which are supposed to draw them backwards in expiration. Neither of these muscles were in action even although the lung on that side was collapsed. The rabbit was in no way dyspnœic and there was therefore no urgency as regards breathing. The second rabbit showed where the power for inspirational movement of the ribs came from, the diaphragm not being there to assist. It also showed that the two intercostals acted together, and all as a group under strong stimulation which recalled the similar action of the occipital muscles. In view of the visible action exhibited by all the inspirational muscles, it would appear that they take very little part in this function and that only when compelled to do so.

From the point of view of anatomy, these results, so far as the intercostals are concerned, are to be expected. The intercostals are unlike any other body-wall muscles in that they are bounded on all sides by thoracic and vertebral skeleton, so that when they act, they exhibit a closed system of forces. In virtue of this, they can only act upon points within that system, which is to say, upon the ribs themselves. These, not being particularly free to move owing to their sternal attachments, are merely pulled upon, giving a stiffening effect to the chest which provides a firm base for the attachments of the greater limb-girdle muscles and the diaphragm. In doing this, the intercostal muscles would actually reduce the capacity of the chest by a trifle: it might be better to say that they prevent undue expansion of the ribs and intercostal spaces in face of the expanding lung. The old idea that the crossed disposition of the fibres of these muscles indicated opposing functions is insupportable as the resultants of their contractions would always be in the lines of the mutual approximation of the ribs.

The electronic investigation of the muscles of respiration by means of a two-beam oscillograph followed very conveniently upon these experiments, and the table overleaf gives the results. These are generally in agreement with those of Robert Gesell (1936) who has worked extensively on this subject.

This method of investigation, probing as it does into the activities of almost individual muscular fibres, has its limitations. It indicates the surge of tonicity without displaying actual contraction, and as tonicity by itself does not perform gross function, the method by itself does not explain very much concerning chest movements. Much the same applies to the findings of Bronk and Ferguson (1935)

1.	Latissimus dorsi	Silent under free and obstructed breathing.
2.	Pectoralis major	Silent except for postural tone on being stretched.
3.	Pectoralis minor	Inspiratory, especially if dead space is increased or breathing obstructed.
4.	Serratus anterior	Inspiratory in quiet breathing; strong expiratory in obstructed breathing.
5.	External intercostals	Inspiratory.
6.	Internal intercostals	Inspiratory, but anterior portion may show expiratory rhythm.
7.	Sterno-mastoid	Silent under both conditions.
8.	Infra-hyoids	Inspiratory.
9.	Scalene	Inspiratory.
10.	Rectus abdominis	Silent unless stretched, giving postural tone.
11.	External oblique	Silent unless stretched, giving postural tone.
12.	Internal oblique	Expiratory rhythm.
13.	Transversus abdominis	Expiratory rhythm somewhat weaker.

who studied by the same means the impulses in nerve fibrils passing to muscles (intercostals). While their results are indisputable, they bear little relation to gross muscular movement which is the purpose of all these experiments and upon which knowledge is required.

These workers in this field have made no mention or allowance for two important features in their experiments. The first is the variable effect of general anæsthesia upon muscular activity. Of this, the anæsthetist is fully aware from experience of fixed doses of drugs upon muscles capable of acting involuntarily or reflexly. This accounts very largely for the variable effects mentioned by Gesell (1936) for the intercostals of eight dogs. Gesell's findings in these dogs have led him to interpret them as individual differences in the breathing pattern. This view does not appear to be tenable, as it seems clear that such a uniform and massive act, as inspiration undoubtedly is, cannot be accomplished by such feeble muscles as the intercostals acting so variably and erratically as he has shown them to do. His

results point much more to the idea that the intercostals are not greatly concerned in the uniform acts of breathing, which have all the indications of concerted action. The second feature that may seriously modify these experiments is the age of the animal, a fact impressed upon the writer in the course of this investigation.

It so happened that for the earlier experiments, rabbits of six months or a little over were used; also a cat used was young although not a kitten. The dogs used were all greyhounds about four years old. The reason for the latter uniformity is that about this age, these animals begin to fail in first-class racing against younger ones and often exhibit bad temper against their rivals. In licenced tracks, these animals are usually destroyed. We were therefore dealing with young dogs which, with the other young adult animals used, showed chest movements at their best.

In a confirmatory experiment at a later date, a fully grown rabbit was asked for and this was produced—an elderly doe. Upon exposure of the chest, this was seen to be quite immobile and was useless for our purpose. Keith (1903) also mentions that the chest of the dog does not move to-and-fro: he evidently has been looking at a dog of unknown age, but certainly not young.

In this we are only seeing in other mammals what we know so well for ourselves concerning athletic prowess. After the mid-twenties it declines, possibly with the declining mobility of the chest which has been developed to the fullest extent to maintain the great demands of a rapidly developing muscular system. When this is attained, the movements of the more specialized individual small muscles such as the levatores costarum and intercostals decline, and with age, this may go on until practically only the diaphragm is left to provide active inspiration, as was found in the elderly rabbit.

The experiments described have therefore yielded visible evidence of the nature of intercostal action and of the activities of certain other muscles concerned with inspiration. No such activity was discernable with regard to expiration although the oscillograph indicated this function for muscles of the abdominal group. Obstruction to the breathing did not have the same effect on the expiratory muscles as upon those of inspiration. This can be verified clinically. A nose-breathing patient under general anæsthesia will exhibit strong inspiratory efforts which include

that of the sterno-mastoid on closing the nares, but there is no corresponding expiratory effort although such powerful muscles exist for this purpose.

The departure from the prevailing view of intercostal action indicated by these experiments is probably best accounted for by a reference to the development of so-called segmented structure in annulous cœlomate forms.

In a paper (Primrose, 1920) dealing with the evolution of the vertebrate endoskeleton, it is pointed out that water is the first substance to be used as an endoskeleton in early animal organization; and in order to make use of this fluid material, especially in vermiform types, a new tissue makes its appearance as a step in evolution. This is the mesoderm of embryology. In the simple cœlomate animals of vermiform shape such as the archannelid, Polygordius, two strips of this tissue grow in between the gut tube and the skin, which latter has an annulated appearance suggestive of that of respirator tubing and for precisely the same purpose (fig. 1). Very soon, a series of cavities begins to appear in each strip from before backwards. The inner or visceral layer applies itself to the gut-tube while the peripheral layer goes on expanding until it comes to lie under the skin, the cavities enlarging along with it. The skeleton of water, or hydrostatic skeleton as it may be called, occupies these cavities and, when fully developed, it appears as a bilateral series of water vertebræ which occupies almost the entire body. The body wall muscles which overlie them differentiate into an outer circular layer which is broken at the septal limits of every segment of skeleton and an inner longitudinal layer which extends the length of the animal as several narrow bands. By their actions upon the incompressible but fluid skeleton, they produce locomotion. The visceral layer of this

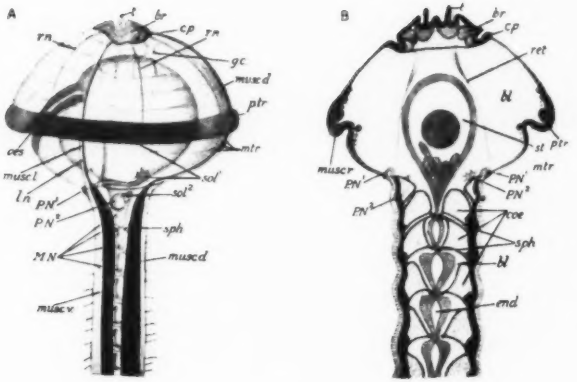

FIG. 1

Diagram showing two stages in the development of Polygordius neapolitanus (after Woltereck) from the spherical trochophore larva. The annulated body grows down from the lower pole of the larva and mesoderm grows as two strips into this between the gut tube and the skin. The mesoderm acquires cavities as seen in the later stage which develop as the locomotor organ.

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mesoderm applies itself to the gut-tube and differentiates the two layers of muscle characteristic of all higher forms for the propulsion of its contents.

When this arrangement of skeleton and muscle is supplied by serially repeated vessels and nerves from common trunks, we recognize, not a condition of segmentation of the body, but the development of an entirely new organ consisting of many serially repeated parts. This arrangement, on account of its extent in occupying nearly all the body, has given rise to the original misconception that the body was itself segmented. This new organ may be called the " locomotor organ " to indicate the principal function it performs for the animal.

This segmentation, which is restricted to the water skeleton, is the solution of a serious problem in cœlomate organization, for water, free in a general cœlomic cavity, would be unmanageable by any kind of muscles and would also constitute a grave danger to the vermiform animal owing to the risk of strangulation of its body during flexion. The arrangement of the body-wall muscles is that required physiologically to operate a fluid but incompressible skeleton of water vertebræ.

The prevertebrate chordates are characterised by the possession of a different kind of endoskeleton. This is the notochord. As an axial skeleton giving support and stability, especially to the dorsal neural tube, it is highly efficient, but its relative lack of flexibility, possibly due to its bulk and turgidity, appears to be a hindrance to the function of locomotion, which requires free flexibility of the trunk as a whole. The forms possessing this skeleton in an unmodified form either do not habitually leave the bottom of their muddy habitat like Amphioxus, or they have to depend upon a parasitic life like the lampreys: yet

they all possess a well-developed locomotor organ which only provides a small degree of free locomotion.

To overcome this difficulty of rigidity, the somites of mesoderm, in which form we now recognize the representative of the primitive locomotor organ, invade the notochord in such a manner as to produce a jointed axial skeleton which retains the original properties but adds the essential feature of flexibility. With this change, and with suitable musculature for a chondro-osseous skeleton, efficient locomotion became accomplished in the various vertebrate groups.

In principle, vertebrate form comes to consist of an elongated cylindrical shape maintained by an axial skeleton and its processes, integrated and overlaid with a muscular body-wall which encloses the viscera, many of which are highly resilient. This tubular body-wall which may show lamination of its muscle into from one to three layers can now only perform one function, namely, compression of the contents of the cavity it encloses, and use must be made of this single function by all the body-wall activities met with in vertebrates. Of these, breathing comes to be an essential function and, with the exception of the mammalia, is based upon compression of the body-wall. This act provides *active expiration* which is the feature of all these animals with the several variations suitable for the needs of each group. The bird in flight shows this principle of respiration most clearly as the lungs are kept passively filled during rapid forward motion and are emptied by abdominal compression.

Compression has been the feature of body-wall musculature since it first appeared in the worms, but it must be noted that these required compression in two directions acting upon an incompressible water skeleton. The muscle

was therefore arranged to do this. In the higher forms with a general cœlomic cavity, general compression only is required, and to effect this, the body-wall musculature largely gives up its segmental or intercostal arrangement, its laminæ arranging their fibres in a decussate manner so as to spread the grouped action of the muscles evenly over the entire cavity. A serial or peristaltic action could have no effect upon a general peritoneal cavity. The Annelida merely present a special instance of body-wall compression which serves to emphasize the fallacy of the idea of metameric segmentation of the body.

The physiological principle of body-wall compression as the fundamental act of breathing has proved efficient for all the vertebrates up to the mammals; but these, with a changed and greatly increased physiology, make demands for oxygen upon the principle of active expiration which cannot be met by existing musculature. A new principle of muscular action and of respiration has to be found to maintain these new conditions.

This change of respiratory principle has been effected by the introduction of a new structure developed in the septum transversum which, until now, has been occupied entirely by the developing heart and its great veins. This septum, originally extra-embryonic in position, is taken into the body of the embryo and it is in this that secondary mesoderm makes its appearance for the purpose of producing a muscular diaphragm which grows beneath the developing heart and lungs. The special function of this new muscular sheet is to provide an improved oxygen supply by means of *active inspiration*, a complete reversal of the physiological principle that has been adequate for all the lower vertebrates. The need for this new muscle is strong evidence that the pre-existing body-wall muscula-

ture, intercostals, etc., is quite unable to provide efficient respiratory function as is so generally believed.

Having reached this point in this brief survey of the chief points in the history of segmented muscle, it will be apparent that this kind of muscle has never played any part in the function of breathing. Segmented muscle is entirely locomotor and may be seen to act in this capacity in the snakes (Owen, 1866). When breathing began to make demands upon the body-wall musculature, this had to give up its segmented character and become laminated as sheets to provide active expiration. While the mammal has retained this it has, in addition, developed a new muscle and a new principle of breathing: it has adapted it also to a chest that still retains the primitive segmented characters, the intrinsic muscles of which, although bilaminar, still only move one rib relatively to another, and that only when most necessary to help other muscles in their inspiratory efforts.

This reference to locomotion brings to light another point, and that is the explanation as to why there are so many large limb-girdle muscles attached to the chest-wall, each anatomically capable of acting upon it, but which, under normal conditions, act from it as a fixed base. The chest, particularly in man, as has been stated, is not meant to move extensively in spite of the arrangement of the thoracic muscles and the elaborate system of diarthrodial joints between it and the vertebral column. These latter are, of course, all parts of the locomotor system, and it will be recalled that flexibility of the axial skeleton is an indispensable requisite for efficient locomotion and that this was the original reason for the segmentation of that skeleton. With few exceptions, this principle is general among vertebrates, the joints being there to allow of

undulating flexibility of the spine during locomotion while the chest remains relatively fixed.

This fixation of the chest is often met with, and to an embarrassing degree, when recourse has to be made to artificial respiration. It is so often apparent that little is achieved by a considerable expenditure of work upon a chest that is not meant to move, especially in the elderly. How very simple is the proceeding when the skeleton is left alone and inflation carried out, either by the conventional anæsthetic apparatus, or, wanting such aid, by mouth-to-mouth inflation (through a gauze filter). Only action upon the flaccid diaphragm will produce satisfactory results in all cases.

From this account of the intercostal muscles, it may be seen that the notion of the paralysis of the intercostals as a sign of deepening anæsthesia (Guedel, 1937) does not exist in fact, as these muscles, even when tense, do not act visibly with clear breathing. The insucking movement seen is phrenic in origin as has already been explained and its diminution is a sign either of weaker action of this muscle as anæsthesia deepens or is due to dilatation of the larger bronchi facilitating respiration. Further, the intercostals are much too deeply situated and are entirely covered by much heavier muscles to show possible activity of their own.

The other important movement of the chest upon which comment may be made is that of circumferential expansion. This occurs passively as a direct result of the angulation of the ribs to the vertebral column. In the case of the second to the fifth inclusive, Keith (1903) points out that the articulations favour the so-called "bucket-handle" movement. It also results from phrenic action. The expansile movement is due to the expansion of the

lung as the descending diaphragm forces it to occupy the enlarging thorax. While the diaphragm is descending, much of the abdominal resistance it encounters facilitates elevation of the chest which the expanding lung exploits fully, even against the tension of the intercostal muscles which limit the "bucket-handle" movement. This is clearly seen in the trans-thoracic operation for gastrectomy. During the exposure, the left side of the chest shows only to-and-fro movement along with the other side. On closure, however, when the lung is finally inflated and the skin wound closed, the expansile movement reappears. During the operation, there is no sign of compensatory movement as breathing is free with adequate oxygenation. Generally, there is no expansile movement of the chest if the lung, for any reason, cannot be filled.

Keith, in his paper on the mechanics of breathing, gives a detailed osteological and arthrological account of the thoracic ribs, and shows that from the sixth to the tenth inclusive they are much more fixed than those above. This difference of anatomy is reflected in the living subject under general anæsthesia when, for any reason, respiration becomes obstructed. The natural heave of the chest and abdomen becomes replaced by an in-drawing of the upper chest and an out-pushing of the abdomen as a result of vigorous phrenic action. The lower thoracic region is observed to be almost at rest by comparison. This see-saw movement is characteristic of completely obstructed breathing.

Of the abdominal muscles, these have been found to contribute a little to normal quiet expiration. This, however, only represents a trifle of their available power for this purpose and almost requires the oscillograph to indicate their activity in this function. The lack of visible effort

has led to the belief that expiration is mostly passive and due to the slight recoil of tonic muscles. Their action in quiet breathing may be seen in cystotomies for removal of the prostate or large calculi in thin elderly subjects. Some of these exhibit a markedly scaphoid abdomen with a chest fixed in inspiration giving a strong concave curve to the recti muscles. When the incision is made for access to the bladder, the separated recti may be seen to be slightly pulled apart by the laminar muscles attached to their sheaths, and this occurs with each expiration. This muscular activity, so often increased by coughing, would appear to be the cause of the difficulty met with in getting cystotomy wounds to close satisfactorily.

Another movement of the abdominal musculature has been observed on a few occasions. This was a wave of contraction seen to pass down the recti abdominis muscles with each expiration. The cases were urgent and for immediate appendicectomy. The first was a well developed young male under light anæsthesia with chloroform which produced very quiet breathing; the second was a similar young female who showed the movement to a lesser degree. This movement is difficult to interpret as it is so rarely seen, but the likelihood is that the local abdominal pain, somewhat severe, reflexly altered the nervous control in this way to avoid the more painful effects of general abdominal compression.

While this treatise has been principally concerned with observations made upon the unconscious subject, it must not be forgotten that breathing is powerfully influenced by the will in the conscious, and that almost any combination of body-wall muscles, particularly the most superficial postural ones, can be brought to bear upon the chest to effect respiration under abnormal conditions. The subject of acute asthma can no longer perform locomotion or do work, but by fixing his limbs he can exert much of his locomotor and prehensile power upon his chest; and in this

effort there are few muscles that cannot be used to some extent, both inspiratory and expiratory. These accessory muscles are brought into action to supplement the diaphragm when its power becomes overtaxed. Their use is abandoned when it is no longer required.

In view of the foregoing, mammalian breathing comes to be in principle a very simple act, although so many interdependent parts are involved in all the variations of its working. An entirely new muscle has been evolved for operating a new principle of respiration and is formed during development for the sole purpose of providing an oxygen supply by active inspiration. Normally, in mammals, there is no equally strong act of expiration in spite of the anatomical arrangement of muscles credited with that function. The great abdominal muscles always associated with expiration provide a little of this, but are much more designed, in the case of man, for doing work under conscious impulse as in the lifting of weights when they, as body compressors, operate strongly with a chest fixed in inspiration by a closed glottis. MacKenzie (1919) points this out. In this connection, it should be noted that the external oblique muscle in the pronograda is a strong flexor of the lumbar part of the trunk for fast locomotion and has no attachment to the ilium; whereas in man, the ilium cuts into the side of this muscle, giving it an extensive attachment as an additional compressor of the abdomen. This power of doing work by compression of the body is a compensation for the loss of much locomotor function in man associated with his erect posture. His body-wall, however, still exhibits the features fundamental to locomotion, but, at the same time, he is permitted to use his locomotor or limb muscles for a variety of other purposes. In common with other mammals, his respiration

represents the latest stage in the evolution of breathing and, in this, the intercostal muscles have come to play a very insignificant part. This conclusion brings the idea developed here concerning these muscles into line with those expressed by Henle, Meissner, von Ebner, and Landois (1911), who assert that " the intercostals are of no great importance in regard to the movement of the ribs; they serve rather to regulate the tension in the intercostal spaces; to impede their retraction by increased negative intrathoracic pressure; and to reinforce them during inspiration." This view is further supported on clinical grounds.

In cases of hemiplegia and other unilateral cerebral lesions normal respiratory movements are not interfered with, but forced respiration does show inequality on the two sides (Walshe, 1951). This would indicate that normal breathing movements are quite involuntary and independent of cerebral control, and that voluntary efforts to intensify breathing are directed from the highest level. It is of interest to note that the recession of costal structure from the anterior parts of the lower six intercostal muscles has allowed these portions to become highly specialized as compressors of the abdomen and, in consequence, they have come under voluntary control and are capable of a high degree of physical training, which cannot be said of the intercostal muscles. More than ever, then, do the intercostal muscles, limited as they are by costal and other skeleton, remain in a primitive physiological state playing a very minor part in the respiratory act, which normally is carried out by the more deeply placed extra-thoracic muscles in association with the diaphragm.

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ANÆSTHESIA AND THE ADRENALS

By MARK SWERDLOW

RECENT work suggests that the adrenals play a major part in the body's reaction to injury and to toxic agents. In view of the broadening field of anæsthesia and the growing number of anæsthetic agents in common use, a knowledge of the effects of these drugs on the adrenal is therefore a matter of some importance.

HISTORICAL

Adrenal Medulla. A considerable number of investigations have been carried out in the past, on a variety of experimental animals with many different anæsthetics, in an attempt to determine the effect of anæsthesia on the adrenal medulla. Three lines of investigation have been followed:

1. Histochemical examination of the chromaffin tissue after various periods of anæsthesia.
2. Assessment of the post-anæsthetic adrenaline content of the suprarenal, either by measured extraction or by the effects of extracts on the frog's eye.
3. Measurement of adrenaline in the adrenal venous blood by means of "caval pocket" experiments.

It has been generally agreed that there is some loss of adrenaline from the suprarenal medulla as the result of anæsthesia. The extent of this depletion and the physiological implications thereof are, however, more controversial. The controversy has largely centred around the work

of Elliott (1912), who found that after anæsthesia there was an increased output of adrenaline with exhaustion of the adrenaline content of the adrenals. Kodama (1923) considers that Elliott's results can be explained by a diminished secretion as well as by an intensified excretion of adrenaline. Elliott's findings, although unconfirmed by Kodama, by Keeton and Ross (1919), or by Shiota (1909), have been substantiated by Fujii (1921), Corbett (1922) and Elmes and Jefferson (1942).

Adrenal Cortex. Relatively little study has been made of the effects of anæsthesia on the adrenal cortex and the results have been varied and inconclusive.

The first workers to study the problem were Delbet, Herrenschmidt and Beauvy (1912). They anæsthetized guinea pigs with chloroform for various lengths of time: they found that progressively longer periods of anæsthesia resulted in increasing lipid in the cortex. This finding was confirmed by Scaglione (1915) and Niemeyer (1921) in later work under similar experimental conditions, although Scaglione noted that the cortical lipid diminished with longer than 5 hours' anæsthesia. Baiocchi (1923) reported that chloroform anæsthesia caused a marked reduction in the lipid content of the dog adrenal, and Manceau (1925) found that chloroform anæsthesia caused a marked reduction in the cholesterol content of the guinea pig adrenal. Abelin (1946) anæsthetized guinea pigs and rats with ether, chloroform, Numal (allyl iso-propyl barbituric acid) and Evipan (soluble hexo-barbitone). He found throughout a marked increase in adrenal cholesterol as determined by extraction. In the following year Ludewig and Chanutin (1947) reported work on the effects of intraperitoneal Nembutal (pentobarbital) on the adrenal of the white rat. They found little change in the adrenal cholesterol and

ascorbic acid content in the first 2½ hours after anæsthesia, after which there was a decrease in both cholesterol and ascorbic acid. Yoffey and Baxter (1949) found that after 10 minutes ether anæsthesia in the white rat, the adrenals showed the picture of the "adaptation" ('alarm') syndrome (see p. 32). Working at the same laboratory, Robinson (1951) has recently confirmed this finding.

METHOD OF PRESENT INVESTIGATION

The majority of the previous workers anæsthetized animals for different fixed periods of time. Variations in the dose of anæsthetic required by individual animals and in the depth of anæsthesia produced were not taken into account, so that changes in the adrenals could not be related to the amount of anæsthetic administered.

In the present investigation the author hoped to determine the histochemical changes in one adrenal of each of a series of male albino rats subjected to equal amounts of anæsthetic per 100 g. body weight, and to assess the degree of recovery by examining the remaining adrenal on killing the animals 24 hours later.

The anæsthetics used were: ether, chloroform, trichlorethylene, pentobarbital (Nembutal) and thiopentone (Pentothal). With each anæsthetic 10 rats received a small total dose administered fractionally; and 10 rats received a larger total dose also administered fractionally. (In addition a small group of rats were each given a very large total dose of ether resulting in approximately 5 hours' anæsthesia.) Individual members of each dosage group received equal amounts of anæsthetic per 100 g. body weight and an even plane of anæsthesia was aimed at throughout. The actual dose used for each dosage group

of each anæsthetic was determined in preliminary trial experiments, and was calculated to produce approximately 1 and 2 hours anæsthesia respectively. Thus comparison could be made between the adrenal changes produced by corresponding doses of different anæsthetics. The size of the small and larger doses of each anæsthetic are shown in Table 1. In view of the difficulty of measuring with any exactitude the moments of entry into and emergence from anæsthesia, the duration of anæsthesia for each animal was timed to the nearest five minutes.

TABLE 1. *Anæsthetic dosage and duration of anæsthesia.*

Anæsthetic	Dosage group	Dose of anæsthetic per 100 g. body wt.	Period of anæsthesia (average of series) in minutes
Ether	Small	2.25 ml.	67.5
	Larger	4.2 ml.	118.5
	'5-hour group'	8.6 ml.	306
Chloroform	Small	0.4 ml.	67
	Larger	0.55 ml.	126
Trichlorethylene	Small	0.35 ml.	68.5
	Larger	0.5 ml.	122
Pentobarbital (1% solution)	Small	0.4 ml.	68
	Larger	0.7 ml.	130.5
Thiopentone (1% solution)	Small	*	65
	Larger	0.9 ml.	128

* The dose/weight basis had to be abandoned in this group because in some cases the minimal anæsthetic dose produced as much as 50 minutes' anæsthesia. Accordingly, with each member of this series only, anæsthesia was maintained for approximately one hour, regardless of dosage.

Experimental Animals. The work was carried out entirely on young adult male albino rats of the Wistar strain, inbred for 7 years under constant conditions. They

were kept in wire-mesh cages in warm dry rooms, and fed with M.R.C. Diet 41, adequate drinking water being available. Before anæsthesia the animals were kept on a bed of sawdust, not more than 10 to a cage. After adrenalectomy they were placed in a clean cage on a bed of wood wool, sawdust being avoided to prevent inhalation of dust whilst still semiconscious. The temperature of the laboratory throughout the period of the experiments was 65 ± 3°F. The weight of the rats used ranged from 110 to 190 g.; 95 per cent weighed between 110 and 155 g. (incl.).

Controls. Two batches, each of 20 rats, were used to provide control data. Those of the first group were killed instantaneously by fracture of the neck; both adrenals were then removed, weighed and placed in formol saline. These were used to demonstrate the normal histological picture of the rat adrenal.

In the second group ("B controls") the right adrenal was removed under brief, open-mask ether anæsthesia. The rats recovered almost immediately and were placed in warm dry cages, food and water being available. Twenty-four hours later they were killed by fracture of the neck and the left adrenal removed. As the only difference between the procedure for these rats and that for the experimental animals was the duration of anæsthesia, the findings for this control group should show the effects of all factors other than anæsthesia itself (e.g. fear and struggling, operative interference).

EXPERIMENTAL TECHNIQUE

Anæsthesia was induced either by intraperitoneal injection (for the barbiturate groups) or by mask and drop bottle. Anæsthesia was then maintained by the periodic addition of small supplementary doses of anæsthetic,

administered when squeezing of the rat's tail resulted in a certain degree of reflex movement. Testing for depth of anæsthesia by squeezing the tail was carried out repeatedly at regular intervals, the interval depending on the particular anæsthetic. (The size of the supplementary doses and the degree of reflex movement adopted as the criterion of depth for each anæsthetic were determined during a number of preliminary trial experiments.) A fairly level plane of anæsthesia was thus maintained, and all the animals receiving one particular anæsthetic were kept at a comparable depth. However, a different standard of depth had to be used for each anæsthetic agent.

With all anæsthetics used, when the rat began to emerge from anæsthesia after having received its total dose, the right adrenal was removed through a longitudinal dorsal incision. Twenty-four hours later the animal was killed by fracture of the neck and the left adrenal removed. Each adrenal was cleaned, weighed and placed in 10 per cent formol saline immediately after removal. Precautions were taken to prevent loss of water from the glands during cleaning and weighing. From each adrenal sections were prepared and stained with Sudan Black B and by Schultz's technique. The former were examined for amount and distribution of cortical lipid and also micrometer measurements were made of the breadth of the total cortex and of the breadth of deeply sudanophil cortex. The Schultz-stained sections were inspected for degree of green-blue cholesterol staining.

RESULTS

Adrenal cholesterol content. No appreciable alteration was found in the cholesterol content of the adrenals of either control or experimental animals. In all anæsthetic

groups both right and left adrenals showed a degree of blue-green staining similar to the normal.

Adrenal lipid content. In the Sudan Black stained sections no material change was found in the lipid content of the cortex, either in comparing corresponding right and left glands or in comparison of each group with the normal. Regardless of dose or nature of anæsthetic used, every section showed a normal distribution of lipid. The normality of the lipid distribution in all groups was confirmed by the cortical measurements made, for in every series the deeply sudanophil band of outer cortex took up a similar proportion of the total cortex to that found in the normal adrenals.

DISCUSSION

The experimental conditions in all the work to date have been so varied that comparison of results is difficult, and, because of species differences, might not be valid. Furthermore, in comparing results we have to contend with the numerous technical difficulties involved in making quantitative assessments from histological material.

The value of the histochemical line of research is at present limited by our inability to explain adequately the variations in adrenal lipid content in terms of effect on function. Thus Flexner and Grollman (1939) consider that increased lipid content in adrenal sections indicates cortical activity, whereas Dosne and Dalton (1941) suggest that loss of lipids is associated with increased activity of the cortex. Abelin (1946) states that increased medullary action is almost certain to occur during anæsthesia, causing at the same time an increased cortical action evidenced by increased steroids, but he produces no experimental evidence to substantiate this opinion. A more recent view

is that of Vogt (1950), who considers that any acceleration of cortical secretion is reflected by an initial depletion of adrenal lipid. This is usually followed by a restoration of lipid, even if the stimulus is prolonged. Thus, as she points out, full lipid stores may be found in the later phases of increased activity as well as in the resting gland, which adds to the difficulty of interpretation.

A somewhat clearer picture of the role of the adrenal during anæsthesia is afforded by a study of recent endocrinological research. Sayers, Sayers, et al. (1944) believe that anæsthesia leads to adreno-corticotropic hormone release and a temporary stimulation of the adrenal cortex. Yoffey and Baxter (1949) and Robinson (1951), after anæsthesia in the rat, claim to have obtained the picture of adrenal stimulation which Selye (1937, 1950) has described as a part of his "general adaptation" syndrome. The picture includes a discharge of chromaffin granules from the medulla, followed by a loss of lipid from the cortex, a fall in ascorbic acid and cholesterol content and a change in colour of the gland to a reddish brown. Selye (1950) himself considers that anæsthetics can cause these changes. Whilst a part of this picture has been seen in some of the researches described above, the cortical changes in particular have been far from constant. In the present work no significant change in the lipid and cholesterol content of the adrenal has been found, and the glands were throughout of a pinkish-yellow colour.

Thus anæsthetics do not in themselves cause a regular stimulation of the adrenal cortex. Indeed once anæsthesia is established the mechanisms involving an adrenal response appear to be depressed. Ronzoni (1950) noted that barbiturate anæsthesia prevents the discharge of adrenal ascorbic acid which normally follows exposure to

cold in the rat. Dury (1948) found that in animals anæsthetized with urethane an adrenal cortical response (in the form of lowered adrenal ascorbic acid) no longer followed the injection of adrenaline. Current research suggests that the injection of adrenaline into a normal animal leads to increased secretion of cortical steroids, via the medium of the A.C.T.H. production in the anterior pituitary. Furthermore, it seems likely that adrenaline is at least partly responsible for normal stimulation of the adrenal cortex, possibly by influencing the hypothalamic effect upon A.C.T.H. release. Dury's observation might therefore be explained by that depression of the brain centres which general anæsthetics bring about. On the other hand, Gellhorn (1950) showed that thiopentone anæsthesia in rats prevented the normal marked lymphopœnia produced by exposure to cold, but not that caused by injection of adrenaline. Thorn (1950) suggests that thiopentone may produce this effect by a local action on the hypothalamus, preventing its response to adrenaline. The lack of response to cold in Gellhorn's experiments is thus presumably due to depression of the sensory pathway or of the higher centres. This is borne out by the fact that Thorn found that the adrenals showed no response to major surgical procedures during spinal anæsthesia.

The important facts would therefore seem to be that anæsthesia does not in itself cause a stimulation of the adrenal cortex, and that it appears to depress the adrenal response to other stimuli mainly by an effect upon the brain centres and the sensory pathway. The appearance of medullary stimulation which anæsthesia can produce (according to several workers) requires further investigation, as do the inconsistencies in the histological findings with regard to the cortex.

From this survey of the previous work in conjunction with the results of the present study, it is considered that future work should be conducted along narrower lines, one anæsthetic only being used for each group of experiments. The rat is not the ideal animal for use in work of this nature, because its adrenals are so small; rabbits or guinea pigs would be better experimental animals, both because their adrenals are larger and therefore afford more sections, and because longer periods of anæsthesia are more easily produced in them. Further research should also include microchemical estimation of the suprarenal contents after anæsthesia. Particularly in view of the difficulty in drawing firm conclusions from histological findings alone, it would be valuable to check these findings by an appropriate technique against the steroid level in samples of adrenal venous blood.

A wide and interesting field remains to be explored, the results of which might not only benefit practical anæsthesia but also contribute to our knowledge of adrenal function.

I am indebted to Messrs. Bengers Ltd., who provided the animals used in the present work and in whose laboratories the investigation was carried out. I also wish to express my gratitude to Dr. Raymond Whitehead for his invaluable assistance in the evaluation of the histological material. Finally, my thanks are due to Dr. F. Fletcher of Benger's Laboratories for much useful advice.

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Brit. J. Anæsth. (1952), **24**, 36.

RESPIRATORY AND CARDIAC CONTROL DURING ENDOTRACHEAL INTUBATION

By MICHAEL JOHNSTONE

MANY papers describing the anæsthesia for endotracheal intubation have been published during the past few years: they indicate that there are considerable differences of opinions as to the safest and best anæsthetic technique for this procedure; and no technique appears to have been consistently satisfactory. The commonest difficulty encountered is laryngo-bronchial spasm which may occur before, during, or immediately after the insertion of the tube; this complication has had tragic consequences (Wylie, 1950). Burstein et al. (1950) have observed that cardiac disturbances, often of a serious degree, may be precipitated by intubation and are particularly liable to occur if bronchial spasm follows intubation or attempts at intubation. The object of the present investigation was to attempt to develop a technique of intubation which could be performed with safety and rapidity on every patient; and which would also be free from respiratory and cardiac complications, particularly in patients with cardiac disease.

The primary reason for administering an anæsthetic prior to intubation is to diminish the activity of the respiratory vago-vagal reflexes and thereby to prevent the occurrence of laryngo-bronchial spasm. An adequate depth of anæsthesia will facilitate the insertion of the endotracheal tube and will prevent traumatization of the larynx. From a strictly pharmacological point of view thiopentone and curare are contra-indicated as neither of

them depress vagal activity unless administered in large doses, the effects of which are not easily reversible. Ether anæsthesia is not always satisfactory as the induction of an adequate depth is often time-consuming. Topical analgesia, combined with light thiopentone anæsthesia, gives good results but cannot be applied to every type of patient or surgical procedure, e.g. oral and dental surgery which may require brisk laryngeal and pharyngeal reflexes on the completion of operations which may last only a few minutes.

Anæsthesia with cyclopropane appeared to me to offer the best possibility of success, as this drug rapidly produces a deep and easily reversible anæsthesia in every patient. Hitherto, cyclopropane has been avoided on account of the disturbances of cardiac rhythm which it may provoke. It has been observed (Johnstone, 1950) that the ventricular arrhythmias associated with cyclopropane anæsthesia—and with thiopentone anæsthesia (Johnstone, 1951a)—were precipitated by the retention of carbon dioxide in the tissues: efficient elimination of carbon dioxide prevented their appearance during anæsthesia, and abolished them when present. It has also been observed (Johnstone, 1951b) that cardiac inhibition follows the inhalation of anæsthetic agents: the irritant anæsthetic vapour stimulates vagal receptors in the air passages, causing reflex inhibition of the heart, with the appearance of bradycardia, auriculo-ventricular nodal rhythms, and cardiac arrest; the removal of the anæsthetic from the air passages is followed by an immediate increase in the pulse-rate with the return of the pacemaker to the sino-auricular node.

In the light of these observations it was assumed that the judicious use of cyclopropane, oxygen, carbon dioxide, and ether might produce a safe and satisfactory method of

anæsthesia for rapid intubation: the cyclopropane was intended to produce rapidly the necessary depth of anæsthesia which would depress sufficiently the laryngeal reflexes; the carbon dioxide was to maintain spontaneous respiration and facilitate the absorption of cyclopropane; the ether was to prevent the escape of ectopic ventricular rhythms; severe degrees of cardiac inhibition would be prevented by the pre-operative administration of atropine. The preliminary investigations to determine the applicability of this hypothesis have been divided into three sections:

(1) The determination of the "mask-off effect" during cyclopropane anæsthesia with carbon dioxide retention;
(2) The effects of ether on ventricular arrhythmias;
(3) The effects of intubation on ventricular arrhythmias.

"MASK-OFF EFFECT" DURING HYPERCAPNIA

A series of 15 patients with normal cardio-vascular systems was selected. Each patient was premedicated with atropine gr. 1/100 (0.65 mg.) and anæsthesia was induced with 0.3 g. of thiopentone injected intravenously. Anæsthesia was then continued with cyclopropane 30 per cent and oxygen in a closed circuit without carbon dioxide adsorption, respiration being assisted gently. Only those patients whose air passages remained perfectly patent, without any evidence of laryngeal or bronchial irritation, were included in this series. After 10 to 15 minutes of anæsthesia the face mask was removed and each patient allowed to breathe air. A portable electro-cardiograph, with visual screen, was under constant observation throughout the induction and maintenance of anæsthesia;

cardiograms were obtained before induction, after the administration of thiopentone, after the period of cyclopropane anæsthesia, and at half-minute intervals after the removal of the mask.

Prior to induction and after the administration of thiopentone, sinus rhythm was present in each patient. During cyclopropane anæsthesia the pulse rates tended to decrease and the pacemakers shifted to the A-V nodes in three patients (fig. 1c). Fifteen seconds after the removal of the mask, sinus tachycardia was present in each patient fig. 1d), and thirty seconds later, ectopic ventricular disturbances had appeared in eleven patients: the ventricular disturbances consisted of multi-focal ventricular tachycardia in two patients, bigeminal rhythms (fig. 1e) of the SA-V type in seven, and isolated ventricular extrasystoles in two. Four minutes after the removal of the mask a regular sinus rhythm was present in each patient.

ETHER AND VENTRICULAR TACHYCARDIA

A second series of 10 patients was selected and each patient was premedicated with atropine gr. 1/100 (0.65 mg.). Ventricular tachycardia was induced in each patient by the inhalation of a mixture of cyclopropane, oxygen, and carbon dioxide in a closed circuit—the arrhythmia usually appeared in approximately 5 minutes. The ether vaporizer was turned on as soon as the arrhythmia appeared, respiration being assisted when necessary. The carbon dioxide absorber was inserted as soon as the effect of ether on the arrhythmia had been noted, and the anæsthetic continued. Only those patients whose air passages remained perfectly patent were included in this series.

[Continued on page 42

FIG. 1

Male, 43 years.

Premedication atropine gr. 1/100.

All Lead 2.

A. Before induction.
B. After thiopentone 0.3 g.
C. After fifteen minutes cyclopropane anæsthesia, with CO_2 retention.
D. Mask off fifteen seconds.
E. Mask off thirty seconds.
F. Mask off four minutes.

FIG. 2

Female, 47 years.

Premedication atropine gr. 1/100.

All Lead 2.

A. Before induction.
B. After five minutes of cyclopropane, oxygen, and carbon dioxide.
C. Ether added ten seconds previously.
D. Ether added twenty seconds previously.
E. Carbon dioxide absorber inserted five minutes previously.

FIG. 3

Female, 34 years.

Premedication atropine gr. 1/100

All Lead 2.

A. Before induction.
B. After thiopentone 0.3 g.
C. After five minutes of cyclopropane, oxygen, and carbon dioxide.
D. After endotracheal intubation, thirty seconds later.
E. On nitrous oxide, oxygen, and ether, forty seconds later.
F. Nitrous oxide, oxygen, and ether, five minutes later.

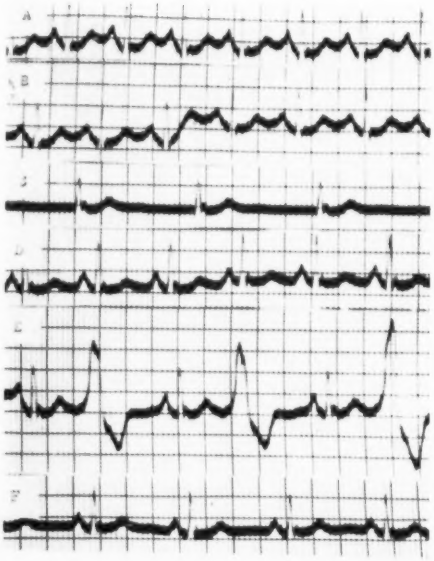

FIG. 1

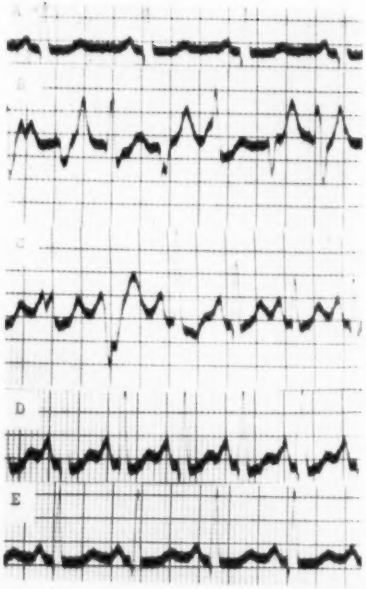

FIG. 2

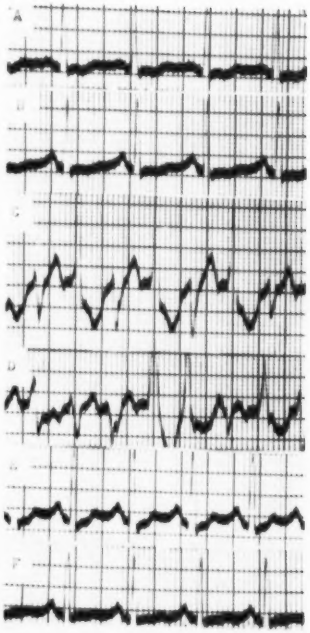

FIG. 3

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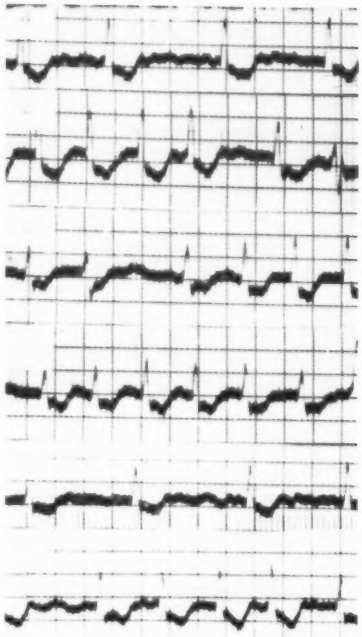

FIG. 4

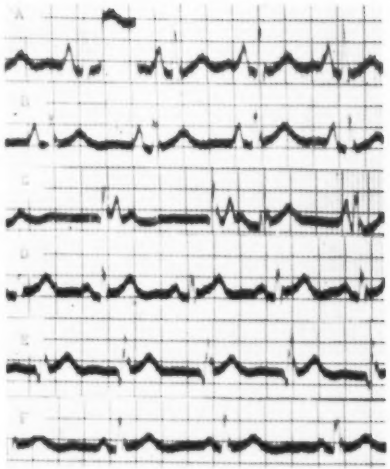

FIG. 5

FIG. 4

Female, 30 years.

Mitral stenosis, with auricular fibrillation and decompensation. Digitalized. Premedication atropine gr. 1/100.

All Lead 2.

A. Before induction.

B. After thiopentone 0.3 g.

C. After six minutes cyclopropane, ether, carbon dioxide, and oxygen.

D. Intubated thirty seconds previously.

E. Seven minutes later, on nitrous oxide, oxygen, and ether anæsthesia.

F. During recovery from anæsthesia.

FIG. 5

Serial tracings illustrating the control of cardiac inhibition during the induction and maintenance of anæsthesia. All Lead 2. Male, 19 years. Congenital pulmonary stenosis, with congestive heart failure. Predemication atropine gr. 1/100.

A. Before induction.

B. After thiopentone 0.3 g.

C. After five minutes of cyclopropane, oxygen, and carbon dioxide; tracing shows auriculo-ventricular dissociation with interference.

D. Thirty seconds later, endotracheal tube inserted; sinus rhythm returned following escape of anæsthetic agent from lungs.

E. Three minutes later, on nitrous oxide, oxygen, and ether anæsthesia; A-V nodal rhythm present.

F. One minute later, sinus rhythm returns following reduction of the ether concentration in inhaled gases.

The administration of ether caused the immediate disappearance of the ventricular arrhythmia in each patient (fig. 2c and 2d), with the return of sinus rhythm in 8 patients and the appearance of A-V nodal rhythm in 2; reduction of the ether concentration caused the return of the pacemakers to the S-A nodes in the latter 2 patients. Momentary removal of the mask a few seconds after the addition of ether was often followed by the reappearance of the arrhythmia which again disappeared immediately after the reapplication of the mask. It was observed, in several patients outside this series, that ether does not abolish the ventricular arrhythmias if its introduction is followed by the appearance of laryngeal spasm; under these circumstances, sinus rhythm does not return until efficient pulmonary ventilation has been re-established. The latter complication may be avoided by introducing the ether gradually and carefully.

ENDOTRACHEAL INTUBATION AND VENTRICULAR ARRHYTHMIA

Ventricular tachycardia was induced in 10 patients, using the technique described above. Endotracheal tubes were then passed and anæsthesia continued with nitrous oxide, oxygen, and ether by the semi-open method. Cardiograms were obtained before induction, after the onset of the ventricular arrhythmia, immediately after the insertion of the tube, and at intervals during the subsequent period of anæsthesia.

It was observed that intubation had no influence on ventricular arrhythmias; a change from bi-focal to multifocal ventricular tachycardia was observed in 2 patients (fig. 3c and 3d), but this change is so frequent during the

course of a ventricular arrhythmia that it cannot be attributed to intubation. As in the previous series, the administration of ether was followed by the immediate disappearance of the ventricular arrhythmia in each patient, with the return of a regular sinus rhythm the rate of which decreased as the carbon dioxide was eliminated.

CARDIAC CONTROL DURING ENDOTRACHEAL INTUBATION

The above observations, in conjunction with those already reported, indicate that the occurrence of ventricular arrhythmias during anæsthesia may be prevented by either of two methods: firstly, by increasing vagal tone by stimulating the pulmo-cardiac reflexes with an irritant vapour; secondly, by ensuring the efficient elimination of carbon dioxide. The second method is the method of choice during the maintenance of anæsthesia as it eliminates the underlying abnormality. Inhibition of the ventricular disturbances by the addition of ether merely masks one of the more obvious signs of hypercapnia and does not influence the basic abnormality (hypercapnia), with the result that the arrhythmias may recur during the recovery phase and severe circulatory collapse ensue.

Abolition of the ventricular arrhythmias by stimulation of the pulmo-cardiac reflexes finds useful application during the induction of anæsthesia. With the judicious use of cyclopropane, oxygen, and carbon dioxide, surgical anæsthesia may be established rapidly in the presence of brisk spontaneous respiration; any tendency towards the occurrence of ventricular ectopic rhythms is offset by adding traces of ether to the inhaled mixture as soon as isolated ventricular ectopic beats appear; isolated ventricular extrasystoles invariably precede the onset of ventri-

cular tachycardia. When the appropriate depth of anæsthesia is obtained, an endotracheal tube may be inserted easily without any disturbance to respiratory or cardiac rhythms. After the insertion of the tube it is advisable to continue anæsthesia for a few minutes with nitrous oxide, oxygen, and ether in order to eliminate carbon dioxide, to prevent the escape of ventricular arrhythmias, and to maintain spontaneous respiration. Anæsthesia is then continued with whatever technique is considered advisable.

Using the above technique, I have recently completed a series of 2,000 intubations. The ages of the patients ranged from 6 weeks to 86 years, and most varieties and degrees of heart disease were included. Intubation was performed with ease and rapidity on every patient; relaxation was complete in the jaw and laryngeal musculature and the vocal cords widely abducted. Reflex bronchiolar spasm following the insertion of the tube was absent in all except the more robust male subjects; in the latter patients the spasm subsided in approximately half a minute. The only disturbances of cardiac rhythm encountered were those due to sudden release of the heart from increased vagal tone as the result of the escape of the anæsthetic from the air passages during the insertion of the tube; these disturbances consisted of sinus tachycardias and ventricular ectopic beats and were easily abolished by the administration of ether or by the elimination of carbon dioxide. Similar disturbances of cardiac rhythm also occurred when bronchospasm followed the insertion of the endotracheal tube into inadequately anæsthetized patients, and disappeared with the return of normal respiratory function.

I have never observed any evidence of reflex cardiac

inhibition following the intubation of atropinized patients. The results obtained in the third section of this investigation indicate that irritation of the trachea fails to inhibit ventricular tachycardia; the inhibition which follows the inhalation of ether appears to occur as the result of stimulation of vagal receptors situated in the distal parts of the lower air passages; a more detailed description of the vagal mechanism has been published (Johnstone, 1951c).

This technique of intubation has been found to be particularly advantageous for patients with heart disease, as the risk of asphyxia due to reflex bronchospasm is eliminated (figs. 4 and 5). It should be performed under electro-cardiographic control and, when dealing with patients with cardiac disease, it should not be attempted until the operator has become thoroughly familiar with the recognition and control of the various cardiac dysfunctions which may be precipitated by the anæsthetic technique. The most dangerous complication associated with the anæsthesia is severe cardiac inhibition or arrest as the result of the rapid introduction of the anæsthetic vapours into the lungs; it is avoided by combining atropine premedication with careful judgment of the anæsthetic concentration which the pulmonary receptors will tolerate. In the early stages of the investigation atropine gr. 1/50th (1.3 mg.) was given pre-operatively to adults; it was soon found that this dose permitted the rapid escape of the ventricular ectopic centres and caused an excessive decrease in the activity of the pulmo-cardiac reflex, thereby rendering more difficult the control of the ventricular arrhythmias. Atropine gr. 1/100 (0.65 mg.) has been found to be satisfactory. Cyclopropane is not without an irritant action on the pulmonary vagal receptors and this action is intensified by manual compression of the rebreathing

bag; this effect often precludes the necessity to add ether to eliminate ventricular arrhythmias.

I have recently modified the method of administering carbon dioxide: the CO_2 inhalations are now commenced immediately before the injection of thiopentone, which is given as soon as the respirations show signs of stimulation —usually about 15 seconds—and discontinued when the thiopentone injection is completed. This procedure has eliminated the respiratory depression and laryngeal spasm which so often follow the administration of thiopentone. Morphine premedication should be avoided as it causes the respiratory centre to become relatively insensitive to small doses of carbon dioxide; and it causes a degree of respiratory depression which may render difficult the rapid attainment of the appropriate depth of anæsthesia.

The important advantage of endotracheal anæsthesia is that the activity of the Kratschmer reflex is eliminated: Kratschmer (1871) observed that the presence of an irritant vapour in the upper air passages caused closure of the larynx and adduction of the vocal cords. I am of the opinion that this reflex is often active during anæsthesia, especially in children, and particularly when one of the more irritant agents is used, and causes marked impairment of respiratory efficiency with retention of carbon dioxide. The latter effect leads to the turbulent diaphragmatic movements so often seen during ether anæsthesia. From the anæsthetist's point of view the diameter of the upper air passages is the distance between the vocal cords. During light anæsthesia the distance between the cords is invariably less than the diameter of the largest endotracheal tube which can be inserted under moderately deep cyclopropane anæsthesia. Therefore, endotracheal intubation ensures efficient and quiet respiration during

anæsthesia and permits the maintenance of anæsthesia with minimal amounts of the agent.

The occurrence of spasm in the jaw musculature following the inhalation of an irritant anæsthetic vapour is probably another manifestation of the Kratschmer reflex and is essentially protective in effect; similar rigidity may also occur during thiopentone anæsthesia if the naso-pharyngeal mucosa is irritated by regurgitated gastric contents, by foreign bodies such as laryngoscopes, or by the inhalation of ether. Cyclopropane, in the concentrations used in clinical anæsthesia, is non-irritant to the naso-pharyngeal receptors and therefore permits the rapid induction of deep anæsthesia with complete relaxation of the jaw and laryngeal musculature throughout its administration.

This investigation has been concerned mainly with oro-tracheal intubation under direct vision, using a Macintosh laryngoscope. The technique is applicable to naso-tracheal intubation and has been used with success on a series of patients with comminuted fractures of the mandible. Naso-tracheal intubation is reserved for surgical procedures which demand its use, as it necessitates the use of tubes of smaller diameter than would be used by the oral route. A size 10 Magill tube has been used for male adults; size 8 oral for female adults; and size 6 nasal for both sexes. The average time for the injection of thiopentone till the commencement of the surgical operation was 6 minutes; discontinuation of the anæsthetic was followed by the return of brisk laryngeal and pharyngeal reflexes within 2 minutes. I have not encountered any complaints referable to laryngeal trauma following intubation; this is undoubtedly due to the fact that the tube is not inserted until the muscles are completely relaxed and the larynx widely dilated.

In dealing with children under the age of 10 years thio-

pentone was not used, anæsthesia being induced and maintained with nitrous oxide, oxygen, and cyclopropane; ether and carbon dioxide were added as required, the indications being similar to those described for adults.

SUMMARY

1. It has been observed that sinus tachycardia and ventricular arrhythmias may be precipitated by the removal of the face-mask from hypercapnic patients anæsthetized with cyclopropane; the mechanism causing these cardiac disturbances appears to be vagal release following the escape of anæsthetic vapours from the lungs; during anæsthesia the arrhythmias are inhibited by stimulation of the pulmo-cardiac reflexes by irritant anæsthetic vapours in the lungs.

2. The ventricular tachycardias associated with cyclopropane anæsthesia in the presence of hypercapnia may be abolished by the addition of ether to the inhaled gaseous mixture. Ether acts by stimulating the pulmo-cardiac reflexes and causing reflex inhibition of the ectopic cardiac contractions.

3. An anæsthetic technique which facilitates rapid endotracheal intubation has been described. It incorporates the use of cyclopropane, oxygen, carbon dioxide, ether, and atropine. The prevention and the control of the cardiac arrhythmias which may be precipitated by the combination of the above drugs have been described.

4. As the result of observations carried out during 2,000 endotracheal intubations it has been suggested that the cardiac arrhythmias which may follow intubation are due either to vagal release following the escape of anæsthetic agents from the lungs, or to partial asphyxia as the

result of bronchospasm precipitated by the insertion of tubes into inadequately anæsthetized patients.

5. It has been observed that it is possible to intubate a patient without causing any respiratory or cardiac upset if moderately deep cyclopropane anæsthesia is used; the use of an electro-cardiograph, with a visual screen, is necessary if cardiac control is to be accurate and safe. The advantages of cyclopropane are its rapid induction and speedy recovery.

6. Some of the disadvantages associated with morphine premedication have been mentioned.

7. The advantages of endotracheal anæsthesia have been described; these appear to be due to the abolition of the Kratschmer reflex by the presence of the endotracheal tube.

I wish to express my gratitude to Mr. E. S. Gawne, F.R.C.S., and to Mr. F. Taylor Monks, M.Sc., F.D.S., R.C.S., for their valuable co-operation during the experimental work.

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A STABLE CARDIO-TACHOMETER OF FAST RESPONSE

By L. MOLYNEUX and E. A. PASK

THE design of a cardio-tachometer falls naturally into two parts. The first part of the instrument should provide a uniform voltage impulse each time the heart beats, and the second should give a visual indication of the rate of occurrence of these impulses. The first part must produce an impulse each time the QRS Complex occurs, but should ignore the "T" wave; simple amplitude selection will not give stable operation, since, in some cases, the amplitude of the "T" wave approaches that of the QRS complex. If, however, a suitably tuned circuit is placed in the amplifier chain, then it may be adjusted to the major frequency component of the QRS complex, so that amplitude discrimination becomes practical (Sturm Wood, 1947).

CIRCUIT DESCRIPTION

First part—A balanced amplifier is arranged to have an unbalanced output which is fed into a parallel "T" rejection filter tuned to mains frequency. The output of this is taken through a variable attenuator, R.15, Box 'A', to a tuned amplifier coupled to a modified Schmidt "trigger" circuit (figs. 1 and 2).

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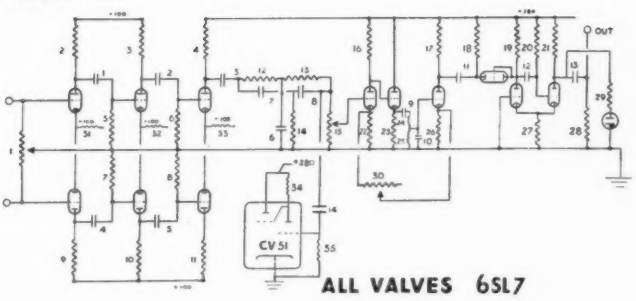

FIG. 1

BOX "A"

R.

1.	250 K	13.	32 K	25.	50 K
2.	220 K	14.	16 K	26.	5.1 K
3.	240 K	15.	500 K	27.	3.6 K
4.	240 K	16.	150 K	28.	1 meg.
5.	1 meg.	17.	150 K	29.	220 K
6.	1 meg.	18.	1 meg.	30.	25 K
7.	1 meg.	19.	150 K	31.	270 K
8.	1 meg.	20.	2.2 meg.	32.	270 K
9.	220 K	21.	150 K	33.	100 K
10.	240 K	22.	5.1 K	34.	1 meg.
11.	100 ohm	23.	100 K	35.	3 meg.
12.	32 K	24.	50 K		

C.

1.	0.1 MF	6.	0.02	11.	0.1
2.	0.1	7.	0.01	12.	0.1
3.	0.1	8.	0.01	13.	0.1
4.	0.1	9.	0.2	14.	0.001
5.	0.1	10.	0.2		

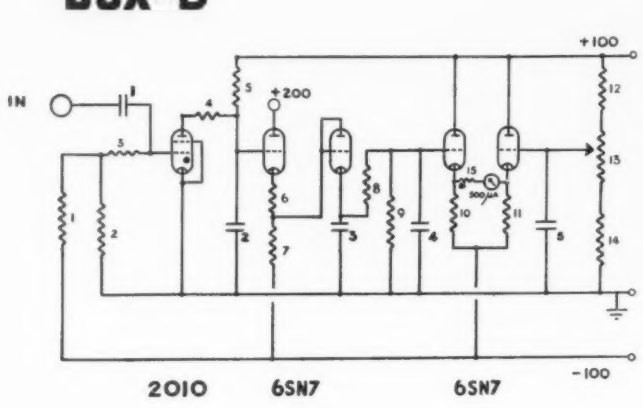

FIG. 2

BOX "B"

R.		C.	
1.	470 K	1.	0.01
2.	47 K	2.	0.5
3.	820 K	3.	0.5
4.	720 ohm	4.	0.01
5.	800 K	5.	0.01
6.	10 K		
7.	75 K		
8.	25 meg.		
9.	2.7 meg.		
10.	75 K		
11.	75 K		
12.	5 K		
13.	50 K		
14.	100 K		
15.	3000 ohm		

ERRATA.

Volume 24, No. 1, January 1952, page 52.

Box B—R.12. 5 K should read R.12. 2.5 meg.

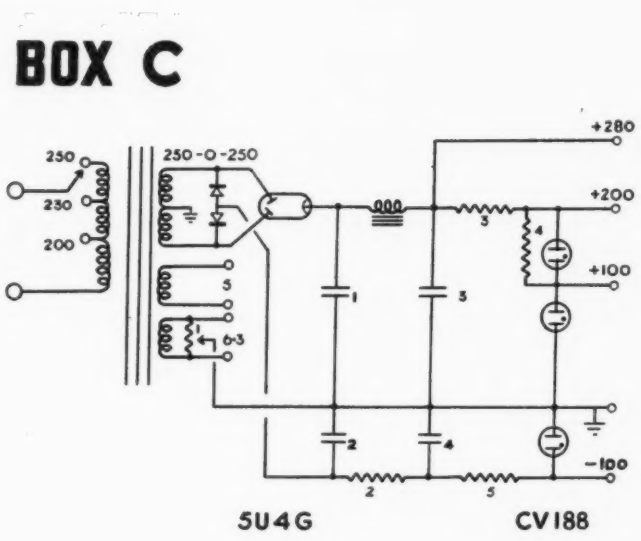

FIG. 3

BOX "C"

R.		C.	
1.	200 ohm	1.	16
2.	5 K	2.	16
3.	10 K	3.	16
4.	20 K	4.	16
5.	15 K		

Second part—The output from the Schmidt "trigger" is used to "fire" a thyratron which is normally biased-off. This discharges a condenser whose voltage is measured by a slow-return peak reading-valve voltmeter, the dial of which is calibrated in pulse rate. (See Appendix.)

Power Pack—This is conventional with simple neon tube stabilization. (Box 'C', fig. 3.)

Construction—The first part of the circuit is contained in a case 10″ × 8″ (IMHOF type 1050), while the second part is housed in a case $2\frac{1}{2}$″ × 7″ × 8″. The power pack is housed in a case similar to the first. The meter is separate.

CALIBRATION

This may best be done with a low frequency oscillator. Adjust R.15 (Box 'B') and R.13 (Box 'B') so that the

full scale deflection = 0 per sec.
half „ „ = 100 per sec.

It will be found that no deflection = approximately 200 per sec.

When this has been done, lock R.15 (Box 'B') and mark the scale at intervals of 5 beats per minute.

OPERATION

It is quite practical to have both the patient and the meter leads 7 yd. long. These long leads have two advantages:

(*a*) The amount of apparatus in the operating theatre is kept to a minimum.

(*b*) The parts of the apparatus which might be capable of causing a flame are kept away from the anæsthetic gases.

Setting-up procedure—The apparatus is switched on and allowed to warm up for 10 minutes, when the indicating meter is adjusted to zero, by R.13 (Box 'B'). Then the amplifier is connected to the patient either by normal E.C.G. connections or by two needles placed in the skin of the chest in suitable positions. The balance control, R.1 (Box 'A'), is adjusted for maximum shadow in the "magic eye", which will partly close each time the QRS complex occurs.

The attenuator control, R.15 (Box 'A'), is adjusted so that the neon bulb flashes once per heart beat. When this has been done, the meter in the operating theatre will display the pulse rate and will continue to do so without further adjustment for the period of the operation, though this may last three hours or more.

ACCURACY AND RESPONSE

Supposing the original calibration to be accurate, the meter reading should be within plus or minus 2 per cent of the true pulse rate.

With the time constants given, the response time is about 0.75 secs. Accordingly, the indicating needle will oscillate slightly on pulse rates lower than 90 a minute, but any cardiac irregularity will cause the needle to swing.

APPENDIX

There are two ways of measuring repetition frequency. The first and direct way is to count the number of impulses occurring in a of counting must be an appreciable fraction of the unit of time which is to be used in designing the frequency rate. Thus, if the given interval of time. For the method to be accurate, the period

result is to be expressed as "beats per minute" one must count over periods of at least ¼ minute. This introduces a delay in registering changes of frequency and brief irregularities of rhythm pass unnoticed.

The other way is to measure the time between two impulses. If a measuring instrument is used, the deflection of which is proportional to this time, a scale upon it calibrated in frequency will be inconvenient, since frequency is proportional to the reciprocal of interval time.

If the measuring device can be made to give a deflection proportional to the reciprocal of interval time, the scale becomes linear in relation to frequency.

These conditions can be approached by allowing a condenser to charge through a resistance from a constant voltage, during the interval between the impulses. The condenser charges in an exponential manner and, if the circuit constants be correctly chosen, only a small and appropriate part of the charging curve is employed. A voltage can then be read from the condenser which is nearly proportional to the reciprocal of the time between impulses.

REFERENCE

Sturm and Wood (1947), *Review of Scientific Instruments*, **18**, 771.

NOTES AND COMMENTS

AN IMPROVED CLAMP FOR THE ANAESTHETIST'S SPHYGMOMANOMETER

This clamp replaces the one now supplied by Messrs. Accoson Ltd. with their anæsthetist's sphygmomanometer. The drawing (figs. 1 and 2) shows the clamp in position on

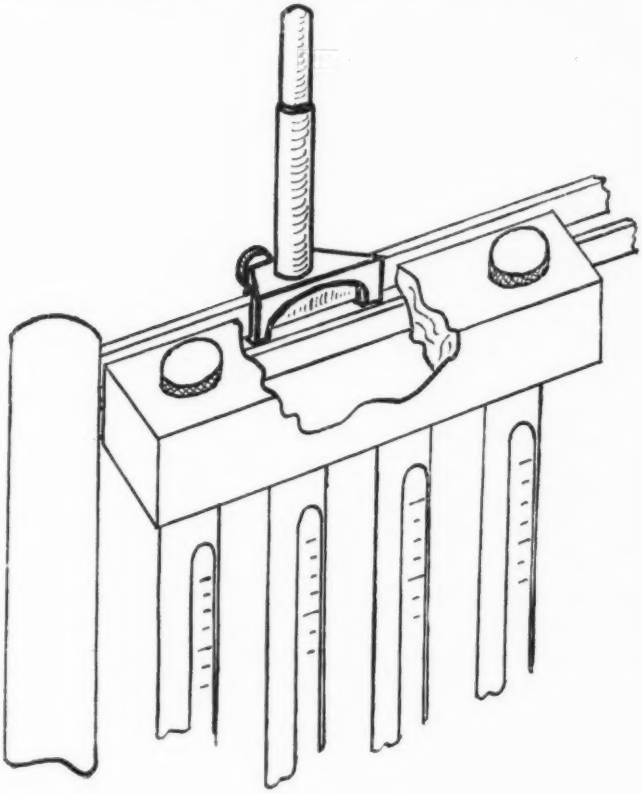

FIG. 1

those rails of the anæsthetic trolley which normally carry the flowmeters and ether and chloroform bottles. In this position the sphygmomanometer is within easy view of the anæsthetist wherever he is, and is at no time in his way.

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Technician, Department of Anæsthesia,
Welsh National School of Medicine

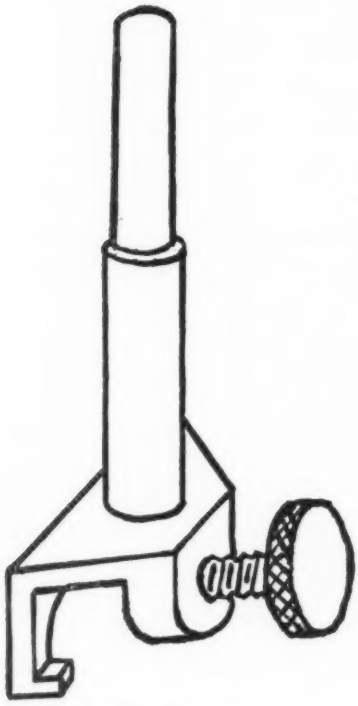

Fig. 2

CORRESPONDENCE

REBREATHING IN A SEMI-CLOSED SYSTEM

Sir,

By the time that your readers have opportunity to consider our comments upon Dr. Domaingue's letter (*Brit. J. Anæsth.*, **23**, 249), they will probably have forgotten all about the original article on which he comments. Do you not think that it would be a good thing when you receive a letter critical of a published article, to offer the authors the chance to comment in a footnote? In this way the authors would be called on to do their duty to your readers and the discussion would be assembled conveniently. With the present arrangement, whereby you do not bring the criticism to the notice of the authors, either before or after you publish it, they might not chance to read it at all and, at best, the discussion is tiresomely delayed because the Journal is published but four times a year.

Some of the words in our article (*Brit. J. Anæsth.*, **23**, 81) were rather careful. We spoke of figures for the flow of fresh gases which must be attained, " before one can be *confident* that rebreathing is eliminated ", and said that if this is desired, " presumably only gas which has *not* come into contact with the alveolar membrane may be permitted to flow back *toward* the bag ". We did not think that exhaled gas " flowed " into the bag and, of course, assumption No. 4 of our Appendix, upon which Dr. Domaingue comments, applies to the system as a whole and not to the bag alone.

The major point of difference is that Dr. Domaingue seems to us to assume that no mixing of gases occurs in the tube. This assumption we considered, but rejected because we believe it always unsafe and often untrue. We sought conditions in which one could be confident that rebreathing does not occur, and therefore proposed that "utilized" air should not *enter the system*, so that the indeterminate degree of mixing could be disregarded.

The mixing in the system is, no doubt, incomplete on occasion, so there is force in Dr. Domaingue's last sentence, provided that the word "*may*" be stressed. We cannot agree with Dr. Domaingue's categorical statement in the last sentence to his penultimate paragraph. We agree that a flow equal to the minute volume will *sometimes* prevent rebreathing in the machine, as, of course, our figure 10 clearly shows.

There is some evidence in figure 4 of our article that the conditions Dr. Domaingue envisages in his penultimate paragraph certainly do not always occur. The figure shows the flow of gases actually measured during a particular type of respiration—in this case "to-and-fro". Immediately after the cessation of backflow, rapid outflow commences. This *rapid* outflow must have been inspiration, and the expiratory valve would be closed. In this case, therefore, there was no opportunity for "expired gases to be driven out by the fresh gas flow from the machine", as Dr. Domaingue suggests. They could only have gone out into the patient.

The matter is very complicated and much must depend upon the actual form, velocity and timing of the flows from the sources. A formula can hardly take a precise account of all these variables, but we think that ours gives figures above which rebreathing can be disregarded and

below which it may be occurring. It would have been possible to set conditions even more exacting.

E. A. PASK
Newcastle

[We are grateful to Professor Pask for his suggestion and will bear it in mind when time permits.—EDITORS.]

ASSOCIATION OF ANÆSTHETISTS OF GREAT BRITAIN AND IRELAND

RESEARCH FUND

The Association of Anæsthetists has funds available to finance research and is prepared to consider applications from anæsthetists for financial assistance in connection with research approved by the Council of the Association for the following purposes:

(*a*) Travel abroad for study.

(*b*) To meet the cost of apparatus, materials or technical assistance.

(*c*) To provide full- or part-time salaries for anæsthetists.

Such applications should be received by the Honorary Secretary of the Association of Anæsthetists, 45 Lincoln's Inn Fields, London, W.C.2, *not later than February 15th*, 1952.

REVIEWS

The Mode of Action of Anæsthetics. By T. A. B. HARRIS, M.B., B.S., D.A., F.F.A.R.C.S., Anæsthetist to Guy's Hospital and to St. Peter's Hospital for Stone. Edinburgh: E. & S. Livingstone Ltd. Pp. 768. 42*s*.

Dr. Harris aims wide of the mark indeed when he suggests that this book fills in the academic lacunæ of undergraduate teaching of anæsthetics. It is doubtful indeed whether even practising anæsthetists will read this book straight through. A mass of information from many sources is brought together, and although the result may make material easily available for reference, few but research workers or doctors are likely to use it. It is probable that there can be few clinical men, to whom the work is addressed, who have had any special training in advanced mathematics, physics or bio-chemistry, and the major part of the text is likely to be nearly unintelligible to them.

A fair sample of the style is "... the adsorption constant, K, would be equivalent to the number of molecules, X, in a mono-molecular layer of the narcotic covering the active surface, multiplied by the area covered by each molecule; this area, assuming that the molecules were spherical in shape, was estimated as the two-thirds power of the molecular volume, Vm, calculated from Lorenz and Lorenz refractive indices. Thus, $K = X(Vm)^{2/3}$. Substituting Freundlich's adsorption formula ..." Yet, oddly enough, the chapters are liberally sprinkled with snippets and cliches such as: "The diaphragm is the most impor-

tant muscle of inspiration. It is a thin musculo-fibrous partition separating the thorax from the abdomen."

These remarks give an idea of the style and manner of presentation. The material content of the book is, nevertheless, admirable. In the first part, the chemical and physical properties of narcotics are discussed in relationship to their narcotic powers. In the second, the same is done for the anæsthetics, a particularly interesting part being a discussion on the relationship of the signs of anæsthesia to the anatomical concept of the selective action of anæsthetics on the central nervous system. In the next section, relaxation is dealt with, while in the last the side effects of anæsthesia are discussed, including, strangely enough considering the title of the book, anæsthetic explosions.

Anyone who has written a book himself will recognize the tremendous labour which has been expended in writing this one. Anyone who has had to teach anæsthetics can only deplore the ponderous style, and hope that in future editions clarification and simplification will be the first task, if, as the author says, his book is written for his clinical colleagues.

Relief of Pain in Childbirth. By W. C. W. NIXON, M.D., F.R.C.S., F.R.C.O.G., and SHILA G. RANSOM, M.R.C.S., L.R.C.P., D.A. Published by Cassell & Co. Ltd. Pp. 109, with 14 illustrations. Price 7*s.* 6*d.*

As the authors state in the Preface, this book shows the result of the opportunity for whole-time observation of a large number of patients. It is written in much detail, and attention is drawn to many points which are usually thought to be of such minor importance as not to be worth mention-

ing. This is especially seen in the careful description of gas-and-air administration, and also in the account of analgesic agents and methods. It is refreshing to read a criticism towards the end of Chapter V on the pain-producing manœuvre of holding back the head. Not only may it tend to rupture muscle fibres which are not visible, but one can speculate on the possible harm to the fœtus.

A suggestion is made on page 3 that in the near future one of the new Trilene inhalers may be passed as safe for use by midwives. Trilene is a drug which has many disadvantages, not to mention dangers, and it would seem a retrograde step for its administration not to be supervised by a doctor.

It is stated on page 54 that it is not thought that patients need or want any analgesic during most of the second stage of labour. Such an opinion is unfortunate, as gas-and-air inhalations, properly timed, are of much assistance then.

This small treatise, which is described as a handbook for the general practitioner, also contains a great deal of practical information for the midwife, and is strongly recommended.